CLINICO-PATHOLOGICAL STUDY OF UROLITHIASIS WITH ANALYSIS OF URINARY STONES IN BUNDELKHAND REGION

THESIS

For

MASTER OF SURGERY

(SURGERY)

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1995 **ASHOK KUMAR GUPTA**

CERTIFICATE

This is to certify that the work of Dr. Ashok Kumar Gupta on "Clinico-pathological study of urolithiasis with analysis of urinary stones in Bundelkhand region" which is being presented by him as thesis for M.S. (Surgery) was conducted in the department of surgery. He is fulfilling the necessary requirements of the stay in the department for the submission of the thesis.

Dated : Dec. 1994

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CERTIFICATE

This is to certify that the work of Dr. Ashok Kumar Gupta on "Clinico-pathological study of urolithiasis with analysis of urinary stones in Bundelkhand region" was conducted under my direct supervision and guidance. I have constantly checked the observations.

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CERTIFICATE

This is to certify that the work of Dr. Ashok Kumar Gupta on " CLINICO-PATHOLOGICAL STUDY OF UROLITHIASIS WITH ANALYSIS OF URINARY STONES IN BUNDELKHAND REGION" had undertaken the facilities of analysis of the urinary stones at CDRI, LUCKNOW under my direct supervision and guidance. I have constantly checked the observations.

DatedDec.1994

(G.K.Patnaik)

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(Ashok Kumar Gupta)

C O N T E N T S

INTRODUCTION

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The problem of urinary stones is not new. These are the man's oldest documented great misfortune. They are notorious for their multi valent out look regarding types, composition and causation that various theories which were conceived to explain the phenomenon could not stand the test of the time.

To investigate long way down in to the subject bring us in notice of a facinating world of opinions that the subject can hardly be compared with other problems.

Earliest evidence of stone is found in predynastic Egyptian mummy dating at about 4800 B.C. The urolilthiasis is known to be prevalent in Hippocratic era in Greece, Celsus era in Rome and in the era of Sushruta in India. Various theories are described but etiopathology is not yet fully established.

No Satisfactory drug is available though many advancements have been made and surgical removal remains the main treatment. But there are recurrence of stone formation in many cases so the problem is still challenging to medical scientists and surgeons. With the help of latest methods medical scientists are trying to understand etiopathogenesis. Medical treatment and Surgeons through Surgery are now capable of preserving maximum number of

nephrons and removing all calculi from Urinary tract.

Renal calculus surgery has emerged from multiplicity of technological and conceptual development.

The problem of urolithiasis is rampant in Bundelkhand region, hence there is need to study the subject. The present study deals with clinicopathological evaluation of disease with analysis of urinary stones.

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REVIEW OF LITERATURE

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Anthropological history provides evidence that urinary calculi existed as long as 7000 years ago or perhaps longer. The speciality of urological surgery was even recognized by Hippocrates, who in his famous oath for the physician stated "I will not cut even for the stone, but leave such procedures to the practitioner's of craft", (Clendening,1942).

Riches (1968) referred to the stone that was found in the pelvis, presumably in the bladder of an Egyptian skeleton estimated to be over 7000 years old. Ellist Smith (1817-1937) described three cases of urinary calculi after examining 900 egyptian mummies (400-1000B.C.). Shattock (1905) described a stone dating about 4200 BC probably of renal origin.

The famous Indian surgeon Susruta, (5th century AD) was the first to describe the surgical techniques in detail in his monumental classic "Chikitsa Samhita". Hippocrates (400-370BC) described that ingestion of muddy river water containing lime caused stone formation (Butt.1956). Ammonios (283-247BC) was the first surgeon to perform lithotomy and primitive lithotrity.

Coludius Galenus (Galen 200AD) preferred use of stone solvents over surgery and recognized risk factor such as heredity, race, climate and diet related with urolithiasis. Albucaris (11th Century) invented instruments for performing lithotomy and for crushing calculi

impacted in urethra.

In the 13th century Preciani and Narcini were famous itinerary lithotomist, while Ron Delet (1500AD) a French physician differentiated between renal and bladder calculus and speculated different factors for their formation. Pieree Franco (15th century) was the first man in Europe to perform suprapubic cystolithotomy.

Dominico and Douglas (1723-1730) were the first person to operate on renal calculi in 1663. Cheselden and Douglas (1723-1730) described high and low operations for lithotomy. John Douglas (1720) published his description on suprapubic operation. John Hunter recognised the similarity between stone formation and calcification. Scheele (1776) isolated uric acid from urine. Wallanston (1776) found uric acid in stones and described the nature of gouty concretions, fusible calculus and mulberry calculus. Francois Morand and Jean Bascilhac (1703-1781) in France introduced head tilt on operating table and a lithotome respectively.

Wangensteen and Coworkers (1969) referred to some of the more famous lithotomists of 17th and 18th centuries. They included Colot, Friar, Jacques, Rau and others. Soon however surgeons trained in antomy and other aspects of medical practice recognized that travelling lithotomists were not as well skilled as their calling might be desired. Many of these well trained individuals, whom Wangensteen classified as professionals began to take an interest in urinary lithiasis. Most of their interest

centered on improvement of techniques for removal of bladder calculi. As an example, Dupuytren who is famous in many areas of medicine and surgery, developed a new type of perineal instrument for removal of bladder calculi (Drach, 1974a).

In 18th century, Alaxander Marcel wrote an assay on the clinical history and medical treatment of calculus. In 1818 the first lithotrite was introduced by Civiale in Paris which was modified subsequently by Heureteloup (1893), Le Roy, Detoilles, Charrure and Hodgon, Sir Henry Thompson and Bigelow of Boston.

Howship recommended administration of alkalies or acids to arrest the development of calculi as did Sir Astley Cooper (cited by Wesson, 1935). Meckelvon Hemsbach (1858), Epstein (1884) and Liesegang (1896) provided evidence of stone formation due to encrustation and inflammation and described presence of concentric lamination of stones. In 1800 Nitze developed cystoscopy which was modernised by Casper in Germany, Ryall in London and Young in Baltimore. As Europeans moved to America, they brought with them their predisposition to form bladder calculi.

Vogel (1970) noted that in America urinary calculus disease was isolated preponderantly to immigrant Europeans. In 1559, Inca reportedly stated that he thought that corn was the factor that prevents the occurrence of urinary calculi in native American Indians (cited by Vogel). Many Indian herbal treatments were adapted in to

the treatment of urinary calculus or gravel disease. Thus Vogel mentioned the use of Haw or Hawthorn tree, Persimmon, Sarsaparilla and decoction of multiple other leaves and twigs as remedies for stone.

Brown (1901) related urinary stones disease with infection while Osborue (1917) related Vitamin A deficiency as a causative factor. Randall (1937) found presence of subepithelial papillary calcification and theorised them as a precursor of renal calculus formation.

Keyser (1923) and Nakano (1923) studied stones by optical crystallography. Prian and Frondel (1947) described a method for the identification of urinary calculi using a petrographic microscope. In a similar manner Jenson (1940) elaborated the X-ray powder differaction technique of Derby Scherrer, the same technique was used by Herring (1962) and Beclar (1967) for analysis of urinary calculi. The use of infrared spectroscopy to determine the composition of urinary calculi was first done by Beischer (1955). Oliver (1966) first described the process of calculus formation within nephron and termed it as an "intranephronic calculoris" while Howard and Thomas (1967-1968) described presence of natural inhibitors of stone formation in urine. Outstanding amount of work in biochemical composition of stones was made by introduction of sophisticated techniques like optical crystallographic analysis polarizing microscopic study, optical analysis. Transmission electron microscopy and activation analysis had definitely equipped us with

thorough understanding of structural complexity of stones.

Spectrochemical analysis of urinary stones were done in 1971 by Hazarika and Balakrishna Rao.

GEOGRAPHICAL DISTRIBUTION AND INCIDENCE :--

Endemic Calculus regions have been identified in areas of southern China in the past (Thomson 1921), Laos (Westermeyer 1971), Coursen(1972), Northern Thailand (Passmore 1953, Halseead 1961, Gershoff, Prien and Chandrapanond 1963), North western India (McCarrison, 1931), the Middle East and Egypt (Hedyet et al,1969, Levy and Falk 1957, Stark 1970, Loutfi, Van Reen and Abdel Hamid 1974), Turkey (Eckstein, 1960) and in some of the other countries fringing the mediterranean.

Boyce and Co-Worker's (1956) and Burkland and Rosenberg (1955) performed an extensive study of the incidence of calculus disease in United States. All investigative groups agreed that the areas of highest incidence are north west, the south east and arid south west. Finlayson (1974) reported other high incidence areas the British, Isles, Scandinavian Countries, Mediterranean countries, Northern India, Pakistan, Northern Australia, Central Europe, portion of the Malayan Peninsula and China.

Curhan G.C. (1994) observed greater risk of Kidney stone in South East of United States men. Lonsdale (1968) and Sutor and Wooley 1970, 1971 and 1974 a) have

reported extensive geographical survey of types of urinary calculi. Sharma and Colleagues (1989) noted the low incidence of struvite stones in India. Hazarika and Colleagues (1974) and Sharma and colleagues noted that upper urinary tract calculi analyzed in India contained mostly calcium oxalate or calcium phosphate (apatite). Nimikin et al (1992) determined prevelance of urolithiasis in Boston.

The first useful study about the calculus disease in India was carried out by Mc-Carrison and his colleagues in 1931. Following an extensive survey they estimated the overall incidence in India as 10 per 1,00,000 population. The regional variations were obvious. He reported 438 per 1,00,000 in Punjab, 266 per 1,00,000 in Hyderabad district, 13 per 1,00,000 in Ahmednagar and only 0.3 per 1,00,000 in Madras.

Rao (1953-1955) in his survey from Mehsana district of Gujrat, obtained a range from 7 to 103 per 1,00,000 of population in different parts of the district. Andersen et al, reporting on bladder calculis cases from Ahmednagar (1951-1957) found an incidence of 8.5 per 1,00,000 population. A clinical study of calculus cases admitted to Ahmednagar hospital from 1951-57 showed usual Indian pattern with a majority of bladder calculi in children having a peak incidence at 5 years and a smaller total number of renal calculi mainly in adults.

Vashi (1959) made a significant report from Manipur area and he stated that 144 cases of bladder

calculi where seen in three years, 83 percent were Hindus and 17 percent were Mohammandans. He implicated dietary habits for this.

The epidemiological data for 1963-1964 collected from three large hospitals in Delhi by Aurora, Taneja and Gupta showed that bladder calculus cases constituted nearly 62% of all cases of urolithiasis.

Andersen (1968) observed that the incidence in India was highest in North West and North East.

Colabawalla (1970) covering 54 localities and found an overall incidence of calculus disease in this country as 50 per 10,000 hospital admissions. He has again pointed to the higher prevalence of bladder calculus in North India as compared to South India.

Aurora and Remlingaswami had collected the figures of urolithiasis from various hospitals in India. However, maximum number of cases of vesical calculus occurring chiefly in male children of poor families have been highlighted by all worker in the field.

ETIOLOGICAL REVIEW :-

CLIMATIC AND SEASONAL FACTORS :-

Prince and Associates (1956,1960), Rivera (1973) and Al-Dabbagh and Fahade (1977) related their observations on seasonal variation in the incidence of urinary calculi to high summer temperatures in south eastern United States. The reported peak incidence of Urolithiasis were in July, August and September.

Yet Cadoff and Co-worker's (1988) found no

difference in summer or winter urinary osmolality between patients who form stone and who do not form stone.

F. Hussain et al (1990) from Bombay noted that metabolically active disease is unrelated to season.

WATER INTAKE :-

Relationship between water intake and urolithiasis depends up on water ingestion lost by perspiration, respiration and mineral or trace element content of the water supply of the region. Rosenberg (1955), Drach (1976), Smith and Boyce (1969), Finlayson (1974), Thomas (1975) and Seftel and Resnick (1990) recommned more water drinking to prevent the recurrence of urolithiasis along with elimination of infection and elimination of urinary obstruction.

Churchill et al (1980), Shuster etal (1982) and Rose and Westbury (1975) noted that excessive water hardness (eg. Calcium sulfate) contributes to calculus disease formation while Juuti and Heinonen (1980) and Sierakowskiet al (1976 and 1978) noted that excessive softness (eg. Sodium Carbonate) causes a greater incidence of calculus disease (Juute and Heinonen 1980, Sierakowspietal 1976 and 1978).

The W.H.O. in its publication: "International Standards for Drinking Water" (1971) has recommended that hardness in water should be expressed in terms of "milliequivalents per litre. The term "soft" and "hard" may then be used as follows:

Soft - Less than 1 mEq (-50 mg) CaCo3/l

Moderately hard - 1 to 3 mEq (50-150 mg.) CaCo3/l

Hard - 3 to 6 mEq (150-300 mg) CaCo3/l

Very hard - Over 6 mEq (over 300 mg.) CaCo3/l

Drinking water should be moderately hard. There is a marked increase in incidence of calculi in hard water areas. Bokina et al (1966) reported that long term use of hard water for drinking is favourable for development of urolithiasis. In 43 calculi analysed by Hazarika and Balkrishna Rao (1974) revealed an unexpected range of various metals in traces, it is seen that these roughly coincide with the content in water of the same elements.

Ioshin (1971) determined 14 elements in drinking water of Bryansk Oblast and in the urinary calculi of person using this water. Muzlevskaia L. S etal (1993) studies the incidence of urolithiasis in relation to hard water drinking.

AGE AND SEX :-

Age and sex also influence the risk of developing urinary calculi. The lower urinary tract calculi show a peak incidence below 10 years of age while the upper urinary tract calculi show a maximum incidence in the second and third decade of life.

Noble(1930) conducted a survey about age incidence in cases of urinary calculi arising in the population of Siam. When these statistics were compared with those of the neighbouring countries of India and China the age incidence appeared similar.

	Under 20 years of age %	Over 50 years of age %
China (Thomson)	49	17
India (Freyer)	45	26
Siam (Noble)	40.5	12

In Siam this disease was most frequent in first decade of life and its frequency gradually decreased towords adult life and old ages.

Hazarika et al.(1974) presented analysis of 101 lower urinary tract calculi of which about 33% were obtained from children(below 15 years) and 67% were from adult patients.

Das (1971) noted occurrence of renal calculi between 21 and 50 years of age. Kabra et al. (1974) revealed that upper urinary tract calculi predominate in the second and fourth decade of life, Assendelft (1900), from Russia, reported only 2 percent females in a series of 630 cases. Andersen et al (1962) reported only 2.9 percent females in his series from Ahmednagar. Das (1971) reported the male to female ratio as 8:1. Kabra et al,(1972) recorded that urethral calculi occur exclusively in males. Mehdiratta et al,(1972) reported 3:1 male to female ratio in upper urinary tract calculi. Hactor et al. found male to female ratio as 4:1.

The Peak age incidence occurs in 3rd to 5th

decade of life and 3:1 male female ratio was reported by Blacklock (1969), Felter and Zimstand (1961), Inada etal (1958), Pak (1987) and Bailey etal (1974), Burkland and rosenberg (1955) and Sutherland and colleagues (1985) reported peak risk of recurrence of calculi at 1.5 and 8 years.

Malek and Kelalis (1975) and Prince and Scardino (1960) reported equal incidence in males and females during childhood.

Yet Vau Aswegen and associates (1989) found that the urinary testosterone concentration as lower in stone former patients than control. Hiao and Richardson (1972) and Finlayson (1974) reported that lower testosterone level protects the children and women from oxalate stones. F. Hussain (1990) reported male/female ratio of 5:1 and high Prevalence of Bladder stones in children of Bombay . Sriboonlue et al (1992) reported male/female ratio of 2:1 in upper urinary tract calculi in Thailand.

HEREDITY AND RACE :-

Numerous Workers have noted rare urinary calculi in the North American Indians, the blacks of Africa and America and the native born Israeli. Gram (1932) and Gold (1951) noted that hereditory capability of forming stones persists while anatomic site has changed. Resnick etal (1968) and Mc Geoan (1960) concluded that urolithiasis requires a polygenic defect.

Dretler etal (1969), Marquardt (1973) and Giugliani(1985) reported that renal tubular acidosis is

associated with recurent episodes of urolithiasis. Trinch-leri A etal (1988) observed higher frequency of stones amongst the first degree relatives of stone patients.

LOCATION OF CALCULI :-

In majority of cases the calculi was removed from the bladder. From the Canton hospital in 1921 Thomson report-ed a series of 3,492 calculi of which 2,962 (85 percent) were bladder calculi. Post (1907) reported a series of 440 bladder and urethral calculi between the years 1880 and 1907 with only 11 kidney and ureter calculi in Beirut. Higgins (1939) noted presence of multiple ureteral calculi in only 7 patients and bilateral calculi in 6 patients, Kretschmer (1942) reported that in 500 ureteral cases , 45.8% calculi were on right side and 51.8% on left side. Drach etal (1986) and Segura etal (1985) reported 55% ureteral calculi on left and 45% on right side. While Thompson (1925) and School (1936) reported equal frequency on both sides. Andersen (1962) collected datas from 39 hospitals in India between (1951)and (1958). The study revealed a proportion of 1109 (85.05%) bladder and 195 (14.95%) kidney calculi. S.P. Tyagi et al.(1974) reported 19 cases (22.35%) from the kidney, 5 cases (5.89%) from the ureter, 56 cases (65.88%) from bladder, 2 cases (2.35%) from urethra and 3 cases (3.55%) spilled out spontaneously from urethra in Dehradun and Aligarh dis-tricts. Fazil Marickar (1977) found 35% renal stones, 23% ureteric stones, 23% bladder, 1% prostatic, 3% urethral and 15% calculus passers in Kerala. Aurora, Gupta and

Taneja (1963-1964) reported from Delhi that bladder calculus cases constituted nearly 62% of all cases of Urolithiasis.

PHYSICAL CHEMISTRY OF CALCULUS FORMATION :-

The calculus is a solid structure in perpetual contact with a solution that contains the dissolved forms of material from which it is made.

There are four major theories in the development of urolithiasis :

1. Super saturation and crystallization theory.
2. Matrix Nucleation theory.
3. Inhibitor Absence theory
4. Epitaxy

Coe et al and Elliot (1973 b), Finlayson (1974, 1978), Resnick and Boyce (1978, Pak (1987), Scott (1975), Vermeulan and Lyon (1968) and William (1974).

SUPER SATURATION/CRYSTALLIZATION THEORY :-

If the increasing amount of substances capable of crystallization are added to pure water at a given pH and temperature, eventually a high enough concentration is reched for the crystals to form. The point at which saturation is reached and crystallization begins is refferred to as the solubility product (s.p.). Many organic molecule such as urea, uric acid, citrate and complex mucoprotein of urine all mutually affect the solubility of other substances.

Finlayson (1974), Nicar and co-workers

(1983), Elliot (1973 A), Welshamn and Mc Geown (1975), Menon and Mahal (1983), Schwillen and co-workers (1982) and Thomas (1988) reported that dificiency of urinary citrate is one of the many factors found in the urine of patients who form stones. Breslan and Pak (1980), Chang Ti (1987),Hodgkinson and Nordin (1967), Thomas (1974), Walton (1965) and Williams (1974 a & b) reported that the amount of substence in urine becomes so great that crystallization occurs inspite of availible solubilizers and inhibitors in stone formers. Uhlmann and chalmar's (1965) noted that it requires energy to push the crystal nucleus to gether.

Drach et al (1972), Finlayson (1978) , Hautmann et al (1980), Randall (1973), Resnick & Boyce (1978) and Lyon (1968) reported that excessive concentration of crystals or spherules may occure in renal papillae either within the tubular lamina or beneath the surface of papillae.

MATRIX NEUCLEATION THEORY :-

Non crystalline protein like matrix of urinary calculi was first described by Anton Von Heyde in 1984 (cited by King 1967). Boyce and colleagues (1969) have persued the role of matrix in stone formation. Allen and Spence (1966) and Mall et al (1975) reported that matrix calculi composed of an average of 65% of matrix by weight. Sutor and O. Flynn (1973) demonstrated that matrix erupt in to previously crystallized mass.

INHIBITORS ABSENCE THEORY :-

Inhibitors may be organic or inorganic.

In organic group Howard & Colleagues (1967), Robertson & Colleagues (1969) and Smith (1989) described peptide inhibitor which enable the urine to hold calcium in solution form. Drach et al (1983) and White et al (1983) reported that some high molecular weight glycoprotein inhibits calcium oxalate crystallization. Nakagawa and associates (1987) described nephro calcium doing the same function. Angel and Resnick (1989) reported importance of organic inhibitors. Citrate was certainly found to be decreased in some patients of urinary calculi that contain calcium or uric acid (Thomas 1988, Welsh Man and Mc Geown 1976).

In in organic inhibitors specially pyrophosphate (Fleisch and Bisaz 1964) affects the calcium phasphate or calcium oxalate system. This is also noted by Bauman and Wacker (1979) and Drach et al (1983).

EPITAXY :-

Hench (1972), Lonsdale (1968 a and b) and Seifert (1967) had received considerable attention about epitaxy. In this one type of crystal may actually be able to grow on the surface of the first eg. calcium oxalate and uric acid do have enough similartties to permit this process of epitaxy.

OCCUPATION :--

Lonsdale (1968b), Blacklock (1969) and Mates (1969) reported that urolithiasis is more common in individuals who have sedentary occuption. Matas (1969), described interesting method for prevention of stone disease. He advised large consumption of beer and butter associated with minimal stone disease.

Robertson and colleagues (1979a and b) studied relationship between occupation, social class and risk of stone formation. Studies from Denmark and Scotland have shown that 17% of surgeons and 11% of anaesthesiologist's have had urinary calculi.

DIET :--

It has been shown that there is influence of diet on the incidence of urolithiasis. Osborne and Mendel (1917) and Mc Carrrison (1931) found that rats fed on diet deficient in fat soluble vitamins, frequently developed urinary calculi. Consumption predominantly cereal, diet has long been blamed (Joly 1929, Mc carrison 1931 and Andersen 1962) .

Passmore (1953) suggested relation betweeen breast feeding and bladder calculi. Andersen (1962) suggested that the high phytic acid and phytase content of grains might combine with calcium in the food and lead to a calcium deficiency to which the body might respond by parathyroid over action. But Neibel (1959) states that only three cases of primary bladder calculi with hyper-

parathyroidism have been reported in comprison with over 700 with calcification in kidney and ureter.

Gershoff and prien (1960) reported that the administration of vetamin B_6 to individuals with a normal B_6 intake has resulted in a decrease in the oxalate excretion and that following the administration of tryptophan in calculus patients, there had been a marked rise in the excretion of oxalates. Deficiency of pyridoxine is known to increase urinaty excretion of oxalic acid (Prien et al 1962) and administration of 10 mg/day of vitamin B greatly decreases the oxalate excretion in normal as well as oxalate calculus formers. Andersen (1972) Suggsted that increase in dietary protein of animal origin might be responsible for the reduction in endemic bladder calculus.

Robertson etal (1979 a and b) studied relationship between intake of animal protein which leads to increase urinary concentration of calcium, oxalate and uric acid. Robertson etal (1979c) suggested an alternative to high animal protein in take for recurrent calcium oxalate stone formers that they should become vegetarian. Hodgkinson (1976) noted excessive ingestion of purines (uric acid), oxalates (Thomas 1975), calcium phosphate and other elements often results in excessive excretion of these components in urine.

Suvachittanont etal (1973) noted that not only diet but also its source may be important. Identical vegetables grown in various part of Thailand contain amount of oxalate that differ by 50 percent or more. High

intake of refined carbohydrate (sugar) will cause an increase urinary calcium in normal subject and much more in stone frormer patients (Liemann, piering , Lennon 1969 and Andersen 1972).

Schwille Po etal (1992) described environmental factors in pathophysiology of recurrent calcium urolithiasis with special reference to diet. Wangood, and Thind etal from India (1991) reported that hte stone former patients lost thier circadian rhythm of urinary citrate excretion. M.Teotia etal (1991) concluded that children with endemic vesical calculus have normal fluoride metabolism. Michelaci YM etal (1992) suggested that in vivo chondroitin sulphate promotes the growth of stones in the urinary tract.

PATHOPHYSIOLOGY OF URINARY OBSTRUCTION ASSOCIATED WITH LITHIASIS :--

Local irriation, decreased GFR and decreased renal plasma flow occurs after partial or complete obstruction (Jones etal (1989) , Barton (1970, Finkle and Smith (1970) and Vanghan and associates (1971 a and b). Gee and Kiviat (1975) observed hypertrophy of rabbit ureteral musculature only afrer 3 days of obstruction. Rose and Colleagues (1975) observed the effects of chronic ureteral obstruction in 24 dogs. Obstruction results in decreased ureteral peristalsis and pressure generation. Vanghan etal (1970, 1971 a and b) , Stecker etal (1971) and Jones and associat4es (1989) observed that relief of obstruction afrer 8 weeks results in rapid increase in

ipsilateral renal blood flow and partial reversal of functional defects.

Schweitzer (1973) concluded that chronic partial obstruction leads to renal damage early in an animal. Production of xanthogranulomatous pyelonephritis is described by Gingell etal (1973). Changes in opposite Kidney in late cases show delay in extraction of pyelographic medium, dilatation of renal pelvis, and a tendency for the contrast to e retained in the pelvis and calyces. Ramlingaswamy and Aurora (1964) reported reactive changes in bladder mucosa in Delhi area.

A striking relation ship with urolithiasis and squamous cell carcinoma of urinary system had been observed by Gilbert etal (1934) , Heggins (1939) and Gahagan and reed (1949) . Holmgreh etal (1989) noted that if infection is the primary cause, therapy is directed eradication of the infection and removal of stone.

URIC ACID LITHIASIS :--

Absence of enzyme urease from human organs was reported by Gutman and yu (1968) amd Yu (1981). Porter (1966),Seegmiller (1973), and Yu and Gut man (1973) noticed that uric acid is kept in solution by diurnal variation in urinary pH and mucoid molecules. Cifuentes etal (1973), Rapaport (1967) , Thomas (1975) , Williams etal (1974 a and Yu 1981) noted that uric acid lithiasis patients have urinary pH below 6. Seegmiller (1973) reported that only one fourth of patients with hyperuricemic gout excrete excess uric acid when on a purine free diet.

Seftel and Resenic (1990) divided uric acid lithiasis in four groups He also advised to rule out Myeloproliferative or neoplastic disease in patients having hyperurecaemia. Drach (1976 a) , Thomas (1975) and Sakhaee and coworker (1983) recommonded that sodium bicatbonate and potassium citrate prevent formation of calcium stone in patients of uric acid lithiasis . Wabner CL etal (1993) observed that orange juice should be beneficial in the control of calcareous and uric acid nephrolithasis.

CYSTINE URINARY LITHIASIS :--

Giugliani etal (1985) suggested that para homozygote and compound heterozyegote (e.g.I/III) usually form cystine stone.

Smith (1974 b) noticed the incidence of cystinuria as one per 20,000. Johansson etal (1980), Koide etal (1982) and Pak (1987) reported utilization of alpha mercaptopropionylglycine in cystinuria and cystine stone disease.

STONE OF URINARY INFECTION OR STRUVITE STONES :--

These are composed of magnesium, ammonium calcium phosphate.Clark and nodrin (1969) indicate in thier introduction to a renal stone research symposium that "This type of stone (struvite) is also probably due to simple precipition from supersaturation solution. Barnhouse (1968), Griffith (1989) and Griffith and Musher (1973) told about physical chemistry in the production of struvite stones.

Hugosson et al (1990) and cox (1974) told infection as the cause of struvite stone to be differentiated from infection as the result of treatmet of previous calculi of different composition Friedlander and Brande(1974) and Griffith et al (1976 b) reported that Proteus, Providentia sp , Pseudomonas, Klebsiella, staphylococcuss (especially S epidermidis) and even mycoplasma were capable of producuing urea . Griffith (1979) and Lerner etal (1989) reported that E coli apparently dose not produce urease.

Mulvaney (1960) and Mulvaney and Hennug (1962) reported hemiacidrin for dissolution of struvite stone, also by Blaivas and co-workers (1975), Jacob and Gittes (1976), and Dretler and P fister (1984a). Palcuer and colleagues (1987), applied these concepts of treatment to out door patients

CALCIUM URINARY LITHIASIS :--

Normal serum calcium concentration in humans averages 9.6 mg% of which 45% is free ionic and 55% is protein bound. Normally renal reabsorption of 98 to 99% of calcium is excellent (col and Park 1988).

Copp (1969), Kleeman et al (1958), Henneman etal (1958), Pak et al (1974)and Preminger etal (1985) reported that when the load to kidney increased eg intestinal hyper absorption or hyperparathyroidism , hypercalciurea results. Marshall and Robertson (1976) reported that the critical factor necessary for precipitation of

calcium phosphate or calcium oxalate in urine is the instantaneous concentrations of the two elements in urine, not the 24 hour concentration.

Various tests to detect hypercalciurea laid down by Drach (1976 b) , Pak et al (1975) , Seftel and Resnick (1990),Gluszek (1988) , Lemann et al, (1969) and Them et al (1978) depicts brief hypercalciurea after carbohydrate or glucose ingestion.

HYPERPARATHYROIDISM :--

It leads to hyperabsorption from gut, bone and increased renal loss of filtered calcium inspite of increased reabsorption (Kleeman et al 1958).Removal of parathyroid gland eliminate the tendency of urinary calculi in affected patients (Harrison and Rose 1973).

Murad et al (1972), Sewart and Broadus (1981) reported that urinary cyclic AMP (cAMP) determinations may aid the diagnosis of hyperparathyroidism.Yendt and Cohanim(1989) advised that elevated serum calcium level and serum parathyroid hormone level to be estimated.Smith (1989) told that final diagnosis of hyperparathyroidism is confirmed only by surgical exploration and demonstration of parathyroid adenoma or hyperplasia.

Other disease that may cause hpercalcaemia and hypercalcuria are idiopathic hypercalceamia (Williams (1974), Sarcoidosis (Ellman and Parfitt 1960), leukaemia, lymphoma, multiple myeloma (sherwood et al 1967),milk alkali syndrome (smith 1974a), myxedema and adrenal insufficiency (Seftel and Resnick 1990 and sherwood 1988).

VITAMIN D INTOXICATION :-

Excessive amounts of vitamin D (more than 1,00,000 units per day) for many months causes hypercalcaemia and hypercalciurea and thus stone formation.

IMMOBILIZATION SYNDROME:-

Total immobilization due to cast, traction, or quadriplegia and space travel may lead to marked loss of calcium from bone with resultant hypercalciurea.

RENAL TUBULAR ACIDOSIS:-

Two forms of the disease exist, type I and type II. In (1936) Butler Wilson et al described a clinical syndrome charactresed by persistent dehydration, hyperchloraemia,hypokalemia, metabolic acidosis, and nephrocalcinosis.Caruana and Buckalew (1988) and Dretler et al (1969) reported that affected patients do not excrete metabolic acid normally. He also described other factors which helps in formation of urinary calculi in these patients.Fellstrom et al(1983) reported that some ofthese patients also have hyperurecemia.

CALCIUM UROLITHIASIS ASSOCIATED WITH HYPEROXALURIA :--

Robertson and co-workers (1969,1972a and b, 1973, 1974, 1976 and 1978)have produced strong evidence that the amount of urinary oxalate at any given time is roughly ten times more important in determining the precipitation of calcium oxalate than the quantity of either calcium or phosphate in urine. Hyper oxaluria may be prim-

ary or congenital, acquired or idopathic.

In primary Hyperoxaluria endogenous formation of excessive amount of oxalate in tissue occures with out any associated pyridoxine deficiency. Dicksteim etal (1973), Fikari et al (1975) and Seftel and Resnick (1990) reported that regional ileitis , colitis and post oprative intestinal bypass have been demonstrated to excrete excessive amount of oxalate in the urine.

Dretler (1973), Koff (1975) , Rattiazzi et al (1975) and Singer et al (1973) reported that Bricker (ileal loop) procedure developed oxalate stone. Ascorbic acid ingestion leads to increased oxalate excretion and megadose of vitamin C therapy has been implicated as risk factor for urinary calculi (Conyer's et al1985). K.V. Prasad (1993) reported that Musa stem Juice was effective in reducing formation and dissolution of preformed calculi in rats , from India.

Patients who have idiopathic stone formation tend to have one or more urinary excess or deficiencies that may promote formation of calcium stone. These include absorptive or renal leak hypercalciuria (Pak et al 1974, 1975 and 1985), hyperoxaluria due to dietary excess or intestinal disease (Smith 1974 a , Thomas 1974), lack of urinary magnesium,Drach (1976 b), Lehmann and Gray (1989) , and excessive uric acid (Coe and Raisen 1973, Coe and Parks 1988).

RARE FORMS OF CALCULI :-

MATRIX CALCULI :-

Boyce (1968), had defined matrix calculi as those stones composed of coagulated mucoids with very little crystalline component. Several clinical reports of these stones have appeared (Allens et al 1966 , Mall et al 1975).

AMMONIUM ACID URATE CALCULI :--

These account for about 0.2% of all stones. Urealytic infection in the presence of excessive uric acid excretion and urinary phosphate deficiency plus the low fluid intake are main causative factor of these stone as found in children of devloping countries (Hsu 1966 and Klohn et al, 1986).

HEREDITARY XANTHINURIA :-

It results in production of xanthine stone. These are rare, radiolucent calculus. No effective therapy exists (Dent and Philpot 1954, Frayha et al 1973).

SILICATE CALCULI:-

Extremely rare in humans (Jakes et al 1973), present in patients who have chronically taken large dose of silicate containing antacids (Haddad et al 1986).

TRIAMTERENE CALCULI:-

Very rare,0.4% of 50,000 calculi (Jorgel et al 1985). Ettinger et al (1980) and Werness et al (1982) reported that triamterene to be used with great caution in

the patients who form these stones.

2.8 HYDROXADENINE CALCULI :-

Very rare, radiolucent occurs in presence of deficient adenine phosphoribosyl transferase . Witten et al (1983), adviced low purine diet and oral allopurinol administration for prevention of these stones.

SPURIOUS OR FAKE CALCULI:-

Sutor and O. Flynn (1973) reported one patient who inserted boiler scale in to her bladder in order to mimic the production of urinary calculi. Sevaral laboratories reported approximately 1 to 2 % of all calculi. In this case it was observed that matrix was not present in the boiler scale.

OTHER THEORIES OF UROLITHIASIS :--

RANDALL'S PLAQUE THEORY :-

Randall (1937) has reported subepithelial plaques on the pyramids which may become denuded of epithelium and deposits can then be laid down on this base from the urine in to pelvis. The hypothesis has been objected recently on the ground that the subepithelial plaque occur with equal frequency in the kidneys of calculus former and non-calculus formors (Nordin and Hodgkin son,1967).

CARR'S THEORY OF BLOCKED LYMPHATICS :-

Carr (1954), postulated that something perhaps a symptom less episode of infection of the urinary tract in early life may cause the valves of lymphatic

draining the renal pelvis to become incompetent. These inefficient lymphatic no longer remove protein and various debris including calcium salts that have leaked in to the interstitial fluid. A primary calculus or calculi can then form in a "Pouch" out side the collecting cysts.

RENAL TUBULAR DAMAGE :--

Ohkawa et al (1964) repoted that hypercalciuria is notorious for damaging renal tubules. Excessive reabsorptive load of calcium (more than 23 mg/day) may lead to calcium deposition in renal tubules and subsequent calculus formation .

AUTO IMMUNE THEORY :--

Seneca et al (1963) assumes that urinary calculus disease is a spontaneous genetically determined, disturbed tolerance which is an auto immune mechanism.

BONE LESIONS :--

In rickets, osteitis deformans, osteomalacia and osteitis fibrosa there is a tendency to the formation of calculus in urinary tract, and this appears to be due to disturbed calcium phosphorus metabolism (Gold stein, 1955).

IRRADIATION :--

In sanatoria people spend the greater part of their time in open air and in what ever sunlight is available. As a result of prolonged exposure to sunlight, vitamin D is synthesized in the skin. Vitamin D causes a increased absorption of calcium and phosphorus from intestine. Thus the tendency to lithiasis is increased.

MISCELLANEOUS FACTORS :--

ECTOPIC OSSEFICATION :--

Dellate et al. (1976) found ectopic ossification in 11.6% of urinary calculi examined by the peterographic procedures and they have suggested that heterotopic ossification might be a pathological factor that induces lithogenesis in some cases.

SULPHONADES :--

The wide spread use of these drugs and particularly sulphapyridine by oral administration in the treatment of various kinds of infection, has produced a number of cases of calculi in upper urinary tract. Concretions are formed principally of acetyl sulphapyridine (Gross, 1939). Urolithiasis with sulphonamides occurs from therapeutic doses.

HORMONES :--

Shattock (1962) advocated two possible mechanisms of the action of hormones on renal concrete formation. Oestrogens and androgens may have a protein anabolic effect analogous to their known effects on the bone marrow matrix. This may produce calculus matrix even in renal tubules. Androgens and oestrogens may decrease the solubility of calcium and phosphates which may collect in the lumen of the tubules.

MAGNESIUM :--

Rizwi (1976) revealed lower levels of urinary magnesium in patient of urolithiasis. Rats kept on low magnesium diet tend to develop calcareous deposits in their tubular lumina (Ko et al 1962). A high magnesium concentration facilitates the super saturation of urine with calcium and oxalate (Kohjler and Uhle, 1966). Thus magnesium acts as inhibitor of calcification.

SODIUM :--

Sodium increases the solubility of urinary calcium salts simply by their presence, High intake of salt tends to decrease the formation of calculi as in Bantu in Africa (Modlin, 1967). Sodium replaces calcium competitively by binding to crystals of hydroxy apatite. Thus sodium is another physiological inhibitor of mineralization.

ANALYSIS OF UROLITHIASIS :-

Burkland and Rosenberg (1955) asked to urologist thier opinion about the importance of stone analysis, many urologists believe that analysis is not important in planning the treatment . It is hoped that this attitude has changed. Dretler (1990) advised that most medical therapy for stone disease is now based on analysis of calculi, and decisions about proper procedures for treatment require knowledge of stone composition.

Many types of analysis of urinary calculi have been proposed , the most common and most practical

type for the clinical laboratory is chemical analysis.

METHODS OF STONE ANALYSIS:-

CHEMICAL

- Qualitative "spot"test
- Quantitative analysis
- Chromatographic and Autoanalyzer methods

OPTICAL

- Binocular dissection microscopy with petro graphic (polarization) microscopy.

INSTRUMENTAL

- Radiographic crystallography
- Infrared spectroscopy
- Thermoanalytic
- Scanning electron microscopy
- Transmission electron microscopy

Hazarika et al (1974 b), Hodgkinson (1969), Laskowski (1965) and Murphy et al (1962) pointed limitation in qualitative analysis for testing urinary calculi Schneider and co-workers (1973) compared chemical x-ray defraction infrared spectroscopy and thermo analytic procedures in analysis of urinary stones.

Prein and Frondel (1947) pointed greater accuracy of optical crystalography and x-ray crystalography over chemical techniques. Importance of analysis has been made by Berman (1975), catalina et al (1970), Lagergren (1955) , Lonsdale (1968 b,1972) and Schmucki et al (1986). Infrared spectroscopic analysis of urinary calculi has been reported by Hazarika and Rao (1974 a), Kistor and

associates (1974) and Takasaki (1971, 1975 and 1989).

INCIDENCE OF TYPE OF UROLITH'S:-

Commonest types of calculi are calcium oxalate, calcium phosphate, or mixtures of the two. The relative proportion of uric acid calculi stays approximately the same the world over except in India. Also we see apprroximately the same proportions of cystine calculi

Some comparative incidence of forms of urinary lithiasis

Percent of stone analysed

	1	2	3	4	5	6
Forms of lithiasis	USA	USA	India	Israel	Japan	G-Britain
Pure calcium oxalate	33	-	86.1	14	17.4	39.4
Mixed calcium oxalate and phosphate	34	73	4.9	64	50.8	20.2
Pure calcium phosphate	6	8	1d,9	-	3.2	13.2
Magnesium amonium phosphate(struvite)	15	9	2.7	12	17.4	15.4
Uric acid	8	7.63	1.2	9	4.4	8
Cystine	3	0.88	0.4	2	1.0	2.8
Artifacts and other	1	1.5	2.6	-	5.8	1.0

1) Prien EL (1974), 2). Herring (1962), 3). Shrama R.N. (1981) 4). Herbeston (1974), 5). Takasaki (1971), 6). Westburys (1974).

Malhotra et al (1968) from New Delhi , Balkishan Rao (1964) from Gwalior , Andersen et al (1963) from Ahamed Nagar reported morphological characterestic and chemical analysis of uroliths.

F. Hussain et al(1990) from Bombay reported all mixed types of stone. S.Korn (1993) observed that no qualitative difference was found between x-ray diffraction and polarization microscopy.

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MATERIAL & METHODS

The present work has been carried out to study the cases of urolithiasis , admitted in the department of surgery of M.L.B. Medical college and Hospital Jhansi from July 1993 to June 1994 . 50 cases of urolithiasis (26 upper urinary tract and 24 from lower urinary tract) have been included for this study.

METHOD OF STUDY :--

A record of cases was made in the following manner :

HISTORY :--

GENERAL INTERROGATION :- Name , Age, Sex, Occupation , Religion, Rural / Urban, Address, Date of admission and discharge.

COMPLAINTS :- All the complaints were recorded with duration in chronological order starting from the time o onset of symptoms till the time of admission to the hospital.

SYMPTOMS :-

PAIN :- (a) Time of onset (b) Site renal angle, upper and outer quadrant of abdomen , groin , suprapubic, tip of penis or perineal (c) Intensity- dull ache or colicky (d) Radiation Loin to groin or tip of penis (e) Relation with micturition (f) Association with vomiting , sweating and strangury.

-Frequency of micturition : normal or increased.

-Haematuria - (a) Duration (b) Gross or microscopic (c) -Initial, total or terminal (d) amount.

-Difficulty in Micturition - (a) duration (B) dribbling of urine (C) sudden stoppage of streem of urine.

-Retention of urine - (a) Duration (b) Acute or chronic.

-Burning in micturition (a) Duration (b) Intensity.

-Fever - (a) Duration (b) with or without rigors.

HISTORY OF PAST ILLNESS :--

Patients were interrogated about any previous episodes of calculus diseases, characterized by pain or other symptoms, passing of calculi or surgery undergone for calculus previously. The symptoms suggestive of tuberculoses, hypertension and diabetes, were inquired into.

FAMILY HISTORY :--

A detailed family history was taken to find out the contribution of hereditary factors in causation of urinary calculi.

PERSONAL HISTORY :--

Patients were subjected to questions to find out addiction to any intoxicant such as smoking and alcoholism.

DIETARY HISTORY :--

A detailed dietary history was recorded to find out the relationship of dietary habit with the occurrence of urinary calculi. Patients were divided into two groups according to the type of food consumed- (a) Vegetarian. (b) Non-vegetarian.

CLINICAL EXAMINATION :--

A detailed clinical examination of the patients was done at the time of admission on following

lines-

(a)GENERAL EXAMINATION : (i) General condition (ii) Malnutrition (iii) Pulse rate (iv) Blood pressure)v) Respiration (vi) Hydration (vii) Pallor (viii) Clinical sign of uraemia.

(b)LOCAL EXAMINATION - To find out (i) Any obstructive lesion in the urinary passage (ii) Renal lump (iii) Distended bladder (iv) Palpable urethral calculus (v) Palpable prostatic calculus (vi) Signs of urinary infection.

(c) SYSTEMIC EXAMINATION :-

INVESTIGATIONS :-

Following investigations were carried out in the patients .

(1) URINE (a) Routine urine examination - albumin, sugar, microscopic (b) Specific gravity and pH (c) Culture and sensitiveity.

(2) BLOOD - (a) Routine Blood examination - TLC , DLC, ESR, Hb% (b) Blood urea (c) Blood sugar (d) serum calcium (e) Serum inorganic phosphorus.

(3) RADIOLOGICAL - (a) Plain skiagram for KUBP region (b) I. V. P. if required. (c) U.S.G.

TREATMENT :-

One of the following operative procedures was adopted in these patients depending upon the type of urinary calculus.

RENAL CALCULUS :-- Pyelolithotomy, Nephrolithotomy, Nephrectomy.

Ureter calculus - Ureterolithotomy,

U.B. Calculus - Cystolithotomy

Uretharal calculus - Urethrolithotomy

EXAMINATION OF URINARY CALCULI :--

Urinary stone sample was collected and sent for physical and chemical examination to C.D.R.I. Lucknow.

MORPHOLOGICAL EXAMINATION :--

Each calculus was subjected to examination for size , shape, surface , colour and weight .

CHEMICAL EXAMINATION :--

Stones of the respective groups were pooled and were chemically anslysed by standard techniques for moisture, ash, calcium, phosphorus, megnesium, oxalate and cholesterol content according to method of Thind and Nath (1969) With some modifications :

(a) MOISTURE CONTENT :--

100 mg of the powdered stone was dried at 110 degree C. to constant weight.

(b) ASH CONTENT :--

The moisture free sample after the above procedure was then ashed at 600 degree C. in muffle furnace for 16 hours in an open platinum crucible. The resultant ash was dissolved in 6 ml of 0.7 N Hcl made to 25 ml volume and used for calcium, phosphate and magnesium determination.

(c) CALCIUM :--

Calcium was estimated in acid ash solution by the method of Ray Sarkar and chauhan (1967).

(d) PHOSPHORUS :--

The method of Fiske and Subba Row (1952) was employed to estimate inorganic phosphorus present in acid ash solution of stone powder.

(e) MAGNESIUM :--

Magnesium content was estimated in acid ash solution of stone powder with reference to standard of magnesium sulphate using PERKIN - ELM ER 1100 B - absorption spectrophotometer

(f) CHOLESTEROL :--

Cholesterol was estimated using 0.1 ml from the stock of 1 mg/ml of urolith powder in 2N H_2SO_4 using the method of Ziatkis etal (1953) with some modifications.

(g) OXALATE :

20 mg of powdered sample was treated with 5 ml of 2N H_2SO_4 and warmed to facilitate the dessolving of oxalate. The filtrate was analysed with Slight modification for oxalate content by the method of Hodgkinson and Williams (1972).

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O B S E R V A T I O N S

OBSERVATION

The present study includes 50 cases of urolithiasis admitted in MLB Medical College Hospital Jhnasi, from July 1993 to June 94.

Patients selection was done as followes :--

-- Only admitted, radiologically proved patients (emergency + Indoor) are taken up for study

-- All age groups

-- Both sexs

-- Upper urinary tract (UUT) calculi 26 (52%)

-- Lower urinary tract (LUT) calculi 24 (48%)

TABLE No. 1
SHOWING HOSPITAL INCIDENCE OF UROLITHIASIS
IN ADMITTED PATIENTS

TOTAL HOSPITAL ADMISSIONS FROM JULY 93 TO JUNE 1994	SURGICAL CASES		UROLITHIASIS CASES		PER 10,00 HOSPITAL ADMISSION
	No.	%	No.	%	
23873	4774	19.99%	298	6.24%	124

TABLE NO. 2
SHOWING SEASONAL VARIATION IN TEMPRATURE OF JHANSI
FROM JULY 1993 - JUNE 1994
AND INCEDENCE OF ADMISSION OF UROLITHIASIS CASES

	TEMPRATURE		TOTAL ADMISSIONS	
	MAX.	MINI.	No.	%
JULY	44.3	21.4	08	16%
AUGUST	42.0	20.2	11	22%
SEPTEMBER	39.0	18.4	4	8%
OCTOBER	38.6	18.0	2	4%
NOVEMBER	31.4	17.0	--	--
DECEMBER	19.6	5.4	--	--
JANUARY	15.4	2.8	1	2%
FEBRUARY	19	3.3	7	14%
MARCH	21.4	6.6	--	--
APRIL	30.2	11.2	4	8%
MAY	39.8	18.0	3	6%
JUNE	46.0	22.0	10	20%

TABLE NO. 3

SHOWING INCIDENCE OF UROLITHIASIS AS PER LOCATION

SITE	NO OF CASES	PERCENTAGE	UNILATARAL		BILATARAL	
			No.	%	No.	%
KIDNEY	21	42%	17	81%	4	19%
URETER	5	10%	4	80%	1	20%
U. B.	22	44%				
URETHRA	2	4%	---	---	---	---
MULTIPLE SITE	---	---	---	---	---	---
TOTAL	50		21		5	

TABLE No. 4

SHOWING AGE DISTRIBUTION OF PATIENTS

TYPE OF CALCULI	TOTAL NO OF CASES	0-10	11-20	21-30	31-40	41-50	51- 60	61-70	71-80
U.U.T. CALCULI	26	2	5	12	3	3	-	1	-
PERCENTAGE		7.69	19.23	46.15	11.53	11.53	--	3.84	-
				65.38%					
L.U.T. CALCULI	24	13	--	1	2	4	3	-	1
PERCENTAGE		54.16	--	4.16	8.32	16.64	12.50		4.16

TABLE NO. 5
SHOWING SEX DISTRIBUTION OF CASES

SITE OF CALCULI	TOTAL NO OF CASES	MALES		FEMALES	
		NO.	%	NO.	%
U.U.T. CALCULI	26	19	73.07%	7	26.93%
L.U.T. CALCULI	24	19	79.16%	5	20.84%
TOTAL	50	38	76%	12	24%

TABLE No. 6

SHOWING RURAL/URBAN DISTRIBUTION OF PATIENTS

TYPE OF CALCULI	TOTAL NO. OF CASES	RURAL		URBAN	
		No.	%	No.	%
U.U.T. CALCULI	26	08	30.76%	18	69.42%
L.U.T. CALCULI	24	17	70.83%	7	29.17%
TOTAL	50	25	50%	25	50%

TABLE No. 7

SHOWING DISTRIBUTION OF PATIENTS AS PER RELIGION

SITE OF CALCULI	TOTAL NO OF CASES	HINDU'S		MUSLIM'S	
		No.	%	No.	%
U.U.T. CALCULI	26	25	96.15%	1	3.85%
L.U.T. CALCULI	24	22	91.66%	2	8.34%
TOTAL	50	47	94%	3	6%

TABLE NO. 8

SHOWING DIETARY HABITS OF THE PATIENTS OF UROLITHIASIS

TYPE OF CALCULI	TOTAL NO. OF CASES	VEGETARIANS		NON VEGETARIANS	
		No.	%	No.	%
U.U.T. CALCULI	26	14	53.85%	12	46.15%
L.U.T. CALCULI	24	18	75%	6	25%
TOTAL	50	32	64%	18	36%

TABLE No. 9

SHOWING INCIDENCE OF SYMPTOMATOLOGY

SYMPTOMS	U.U.T CALCULI CASES		L.U.T. CALCULI CASES	
	No.	%	No.	%
PAIN	26	100%	24	100%
HAEMATURIA	6	23.07%	4	16.66%
INCREASED FREQUENCY OF MICTURITION	7	26.92%	22	91.66%
RETANTION OF URINE	--	--	3	12.50%
BURNING DURING MICTURITION	20	76.92%	16	66.66%
FEVER	5	19.23%	4	16.66%
HISTORY OF RECURRENT STONE	2	7.70%		
HISTORY OF STONE IN FAMILY	1	3.80%	--	--
SIGN AND SYMPTOM OF RENAL FAILURE	--	--	--	--

TABLE No. 10

SHOWING INCIDENCE OF PHYSICAL SIGN

S. No.	PHYSICAL SIGN	UUT CALCULI CASES		LUT CALCULI CASES	
		NO.	%	NO.	%
1	MALNUTRITION	2	7.69%	8	33.33%
2	ANAEMIA	--	--	4	16.66%
3	RENAL LUMP	2	7.69%	--	--
4	DISTENDED U BLADDER	--	--	2	8.33%
5	PALPABLE URETHRAL CALCULI	--	--	2	8.33%
6	SIGNS OF URINERY INFECTION	10	38.46%	11	45.83%

TABLE No. 11

SHOWING INCIDENCE OF ASSOCIATED DISEASE

S.No.	ASSOCIATED DISEASE	NO.	PERCENTAGE
1	BPH	4	8%
2	KOCHS LUNG	3	6%
3	URNIARY TRACT OBTRUCTION	---	---
4	D.M.	---	---
5	HYPERTENTION	---	---
6	C.H.F.	---	---
7	ACID PEPTIC DISEASE	---	---
8	FEATURES OF SECENDARY HYPERPARATHYROIDISM	---	---
9	RENAL TUBERCULOSIS	---	---
10	HIGH ALKALI INTAKE	---	---
11	DIARRHOEAL DISEASES	---	---
12	RECUMBENCY	---	---

TABLE No. 12

SHOWING URINE EXAMINATION FINDING

	NO OF PATIENTS	PERCENTAGE
ALBUMINURIA	10	20%
GLYCOSURIA	---	---
pH ACIDIC	33	66%
pH ALKALINE	17	34%
MICROSCIPIC		
- PUS CELL	30	60%
- R.B.C.'S	11	22%
- CRYSTALS	18	36%
- CASTS	9	18%

URINE SPECIFIC GRAVITY :--

The specific gravity of the urine was normal in all the cases ranging between 1.012 to 1.0115.

BLOOD EXAMINATION

TLC and DLC :-
The leucocytosis was present in 4 cases of urinary calculi,
ESR :-
It was significantly raised in 3 cases and these are having tuber culosis of lung

HB% :-
4 cases were having heamoglobin level less than 10gm% (LUT calcui cases)
Blood Urea.
Blood urea level was more than 40gm% in 3 cases. The blood urea level was found to be higher in UUT calculi cases (6%).
Blood Sugar :-
Blood Sugar level was found normal in all cases.

TABLE No. 13
SHOWING BACTERIOLOGICAL STUDY OF URINE

TYPE OF BACTERIA	U.U.T. CALCULI 26 CASES		L.U.T. CALCULI 24 CASES	
	No.	%	No.	%
STERILE	20	76.92	9	37.50
E. COLI	3	11.53	8	33.33
KLEBSIELLA	---	---	1	4.16
PROTEUS	---	---	---	---
PSEUDOMONAS	1	3.85	2	8.33
MIXED	2	7.70	4	16.66

TABLE No. 14

SHOWING RADIOLOGICAL FINDING OF PLANE
X RAY ABDOMEN K.U.B.P. REGION

SITE	TOTAL NO OF CASES	UNILATARAL NO.	%	RT No.	%	LT No.	%	BILATARAL No.	%	SINGLE No.	%	MULTIPLE No.	%
KIDNEY	21	17	81	12	70	5	30	4	19	12	57	9	43
URETER	5	4	80	1	25	3	75	1	20	5	100	---	---
U.B	22	-	---	-	-	---	---	-	---	19	86.36	3	13.64
URETHRA	2	-	---	-	-	-	---	-	---	---	---	---	---

TABLE No. 15
SHOWING RADIOLOGICAL FINDINGS ON IVP

S. No.	FINDINGS	NO	PERCENTAGE
1.	HYDRONEPHROTIC CHANGES WITH DELAYED EXCRETION	4	8%
2.	DELAYED EXCRETION WITH OUT HYDRONEPHROSIS	4	8%
3.	NON FUNCTIONING KIDNEY WITH MULTIPLE RENAL CALCULI	1	2%
4.	ECTOPIC KIDNEY (HORSE SHOE SHAPE)	1	2%
5.	NORMAL EXCRETION	28	56%

– Rest of the patients were subjected for U.S.G. examination showing normal renal parenchymal texure with calculi and normal pelvicalceleal system.

TABLE No. 16

SHOWING SERUM CALCIUM LEVEL

S.No.	SERUM CALCIUM LEVEL IN MG%	U.U.T. CALCULI 26 CASES		L.U.T. CALCULI 24 CASES	
		No.	%	No.	%
1.	8–9	2	7.70%	2	8.34%
2.	9–10	20	77.00%	18	75%
3.	10–11	4	15.30%	4	16.66%

TABLE No. 17

SHOWING SERUM INORGANIC PHOSPHORUS LEVEL

SERUM INORGANIC PHOSPHORUS LEVEL IN MG%	U.U.T. CALCULI 26 CASES		L.U.T. CALCULI 24 CASES	
	No.	%	No.	%
2.5 -- 3	2	7.7%	5	20.83%
3 -- 3.5	2	7.7%	4	16.67%
3.5 -- 4	5	19.20%	3	12.50%
4 -- 4.5	15	57.70%	10	41.67%
4.5 -- 5	2	7.70%	2	8.33%

TABLE No. 18

SHOWING MANAGEMENT OF CASES

NAME OF OPERATION		NO.	PERCENTAGE	POSTOPRATIV COMPLICATION	
				No.	%
KIDNEY	NEPHROLITHOTOMY	18	36%	2	4%
	PYELOLITHOTOMY	2	4%		
	NEPHRECTOMY	1	2%		
URETER	URETEROLITHOTOMY	5	10%		
U.B.	CYSTOLITHOTOMY	22	44%		
URETHRA	URETHROLITHOTOMY	2	4%		

MORPHOLOGY OF CALCULI :--

The Urinary calculi were of different size, shape, weight and colour. Size varries from less than 2 cm. to more than 6 cm. with in largest diemeter.

Most of the kidney calculi were rounded and ovoid, bladder calculi oval and ureteric were oblang in shape.

Outer surface was irregular in 65.83% , smooth in 23%, coarse granular in 6.97% and mulberry like in 5.20% cases.

The weight of urinary calculi ranged in between less than 4gm. to more than 96 gm.

Most of kidney calculi were dark brown to black in colour, ureteric, skin to brown colour and vesical chalky white in colour.

TABLE No. 19

SHOWING MOISTURE CONTENT OF URINARY CALCULI

MOISTURE CONTENT IN MG%	U.U.T. CALCULI 26 CASES		L.U.T. CALCULI 24 CASES	
	No.	%	No.	%
0 -- 5	4	15.38	5	20.83
6 -- 10	12	46.15	12	50.00
11 -- 15	3	11.53	3	12.50
16 -- 20	2	7.69	3	12.50
21 -- 25	2	7.69	1	4.17
> 26	3	11.53	0	0

TABLE No. 20

SHOWING ASH CONTENT OF URINARY CALCULI

ASH CONTENT IN MG%	U.U.T. CALCULI 26 CASES		L.U.T. CALCULI 24 CASES	
	No.	%	No.	%
0 -- 25	0	---	1	4.16
26 -- 50	6	23.70	4	16.67
51 -- 75	15	57.70	10	41.67
>76	5	19.23	9	37.50

TABLE No. 21

SHOWING PHOSPHATE CONTENT OF URINARY CALCULI

PHOSPHATE CONTENT IN MG%	U.U.T. CALCULI 26 CASES				L.U.T. CALCULI 24 CASES			
	MALES		FEMALES		MALES		FEMALES	
	NO.	%	No.	%	No.	%	No.	%
0 -- 4	8	30.77	6	23.07	15	62.50	4	16.66
5 -- 8	4	15.38	--	--	2	8.33	1	4.17
9 -- 12	1	3.85	--	--	1	4.17	--	--
13 -- 16	2	7.70	--	--	--	--	--	--
17 -- 20	1	3.85	--	--	--	--	--	--
> 20	3	11.53	1	3.85	1	4.17	--	--

TABLE No. 22

SHOWING CALCIUM CONTENT OF URINARY CALCULI

CALCIUM CONTENT IN MG%	U.U.T. CALCULI 26 CASES				L.U.T. CALCULI 24 CASES			
	MALES		FEMALES		MALES		FEMALES	
	No.	%	No.	%	No.	%	No.	%
0 -- 4	---	--	--	---	--	--	--	--
5 -- 8	--	--	--	---	--	--	--	--
9 -- 12	15	57.70	5	19.33	18	75	3	12.50
> 12	4	15.38	2	7.69	1	4.17	2	8.33

TABLE No. 23

SHOWING MAGNESIUM CONTENT OF URINARY CALCULI

MAGNESIUM CONTENT IN MG%	U.U.T. CALCULI 26 CASES				L.U.T. CALCULI 24 CASES			
	MALES		FEMALES		MALES		FEMALES	
	No.	%	No.	%	No.	%	No.	%
0 -- 1	13	50	6	23.07	14	58.33	5	20.83
1.1 -- 2	3	11.53	--	--	2	8.33	--	--
2.1 -- 3	1	3.85	--	--	2	8.33	--	--
3.1 -- 4	--	--	1	3.85	--	--	-	--
>4	2	7.69	--	--	1	4.17	--	--

TABLE NO. 24

SHOWING OXALATE CONTENT OF URINARY CALCULI

OXALATE CONTENT IN MG%	U.U.T. CALCULI 26 CASES				L.U.T. CALCULI 24 CASES			
	MALES		FEMALES		MALES		FEMALES	
	No.	%	No.	%	No.	%	No.	%
0 -- 0.5	--	--	--	--	--	--	--	--
0.6 -- 1	6	23.07	--	--	4	16.66	2	8.33
1.1 -- 1.5	-	--	1	3.85	7	29.16	2	8.33
1.6 -- 2	6	23.07	4	15.38	4	16.16	-	--
2.1 -- 2.5	6	23.07	2	7.70	4	16.16	1	4.16
2.6 -- 3.0	1	3.85	-	-	-	--	-	--

TABLE No. 25

SHOWING CHOLESTROL CONTENT OF URINARY CALCULI

CHOLESTROL CONTENT IN MG%	U.U.T. CALCULI 26 CASES				L.U.T. CALCULI 24 CASES			
	MALES		FEMALES		MALES		FEMALES	
	No.	%	No.	%	No.	%	No.	%
0 --0.25	11	42.30	4	15.38	16	66.66	5	20.83
0.26 --0.5	3	11.53	3	11.53	3	12.50	--	--
0.51 --0.75	3	11.53	-	--	-	--	--	--
0.76 -- 1	2	7.69	--	--	-	--	---	--

TABLE No. 26

SHOWING COMPARATIVE INCIDENCE OF FORMS OF
URINARY LITHIASIS BY VARIOUS AUTHORS
OF DIFFERENT COUNTRIES

FORMS OF LITHIASIS	PERCENT OF STONE ANALYSED					
	USA (1)	USA (2)	INDIA (3)	ISRAEL (4)	JAPAN (5)	G. BRITAIN (6)
PURE CALCIUM OXALATE	33	--	86.1	14	17.4	39.4
MIXED CALCIUM OXALATE AND PHOSPHATE	34	73	4.9	64	50.8	20.2
PURE CALCIUM PHOSPHATE	6	8	1.9	--	3.2	13.2
MAGNESIUM AMONIUM PHOSPHATE (STRUVITE)	15	9	2.7	12	17.4	15.4
URIC ACID	8	7.63	1.2	9	4.4	8
CYSTINE	3	0.88	0.4	2	1.0	2.8
ARTIFACTS AND OTHERS	1	1.5	2.6	--	5.8	1.0

1). PRIEN E.L. (1974), 2). HERRING (1962), 3). SHARMA R. N. (1989), 4). HERBESTON (1974), 5). TAKASAKI (1971), 6). WESTBURYS (1974).

TABLE No.27

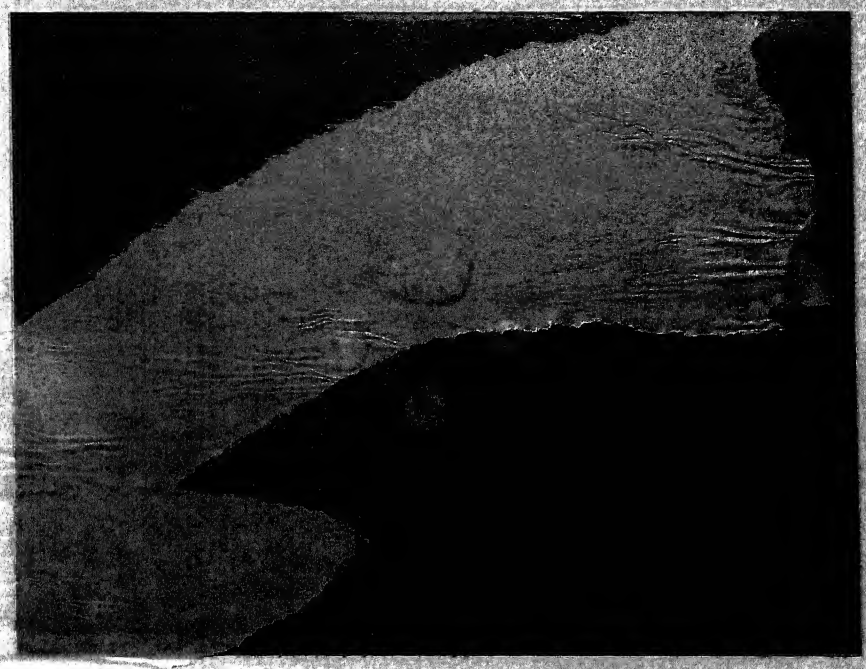
SHOWING COMPARATIVE STUDY OF INCIDENCE OF DIFFERENT CONSTITUENT OF URINARY CALCULI IN INDIA

	1	2	3	4	5	6	7	8	9	10
RADICAL CONSTIUENT	CHANDIGARH 1976	DELHI 1969	GWALIOR 1964	JHANSI PRESENT STUDY	RAJASTHAN 1972	BOMBAY 1990	AHAMDABAD 1960	AHAMAD NAGAR 1963	KERLA 1976	TRIVENDRAM 1988
CALCIUM	97.6%	100%	99.5%	100%	91.5%	96.1%	100%	90%	99%	95.2%
OXALATE	92.8%	96.0%	61.7%	100%	68.5%	99%	38.9%	96.7%	100%	81.5%
PHOSPHATE	75.4%	34%	91.5%	100%	54.8%	97%	100%	100%	76%	96.5%
URATE	32.4%	38.25%	77.7%	--	58.5%	77.5%	76.1%	10%	6%	14.3%
MAGNESIUM	28.8%	9.35%	10.6%	100%	35.0%	61.1%	28.5%	26.6%	44%	89.7%
AMMONIUM	25.2%	25.5%	81.3%	--	56.5%	88.8%	90.4%	16.6%	60%	12%
CARBONATE	20.1%	3.4%	0.88%	--	23%	41.7%	15.9%	--	7%	4.5%
FIBRIN	1.1%	--	18.2%	--	--	--	--	--	--	1.3%
CYSTINE	---	--	--	--	--	--	1.5%	--	--	--
CHOLESTROL	---	--	--	100%	--	--	--	--	--	--

1. S.K. Thind et al (1976),
2. Singh et al (1969)
3. Rao et al (1964)
4. Present study
5. Kabra et al (1972)
6. F Hussain et al (1990)
7. Parikh and Shah (1960)
8. Anderasen et al (1963)
9. F Marickar et al (1976)
10. P Hyacinth, F Marickar et al (1988)

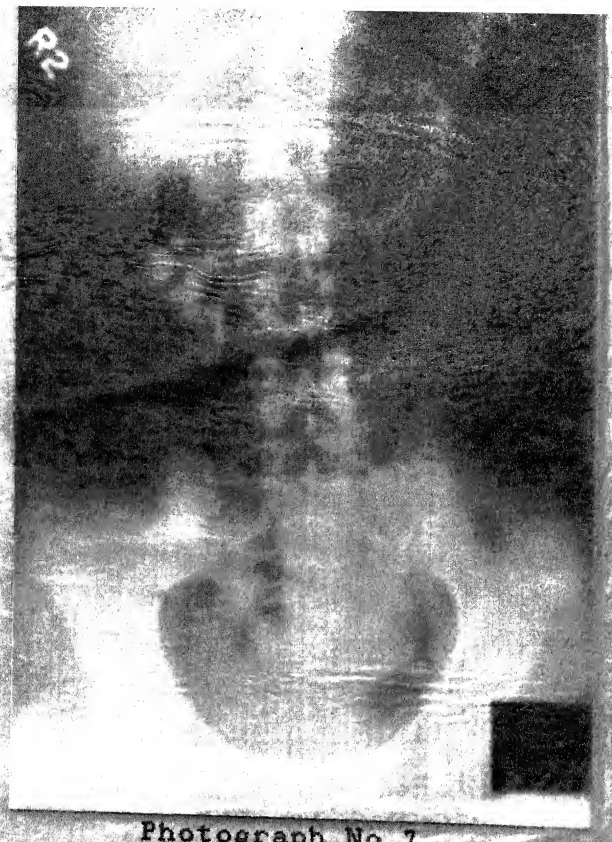
Photograph No. 1
Showing urinary calculi
(a) Renal calculi(b)Ureteric calculi(c)Urethral calculi
(d) Bladder calculi

Photograph No.2
Showing large baldder calculus

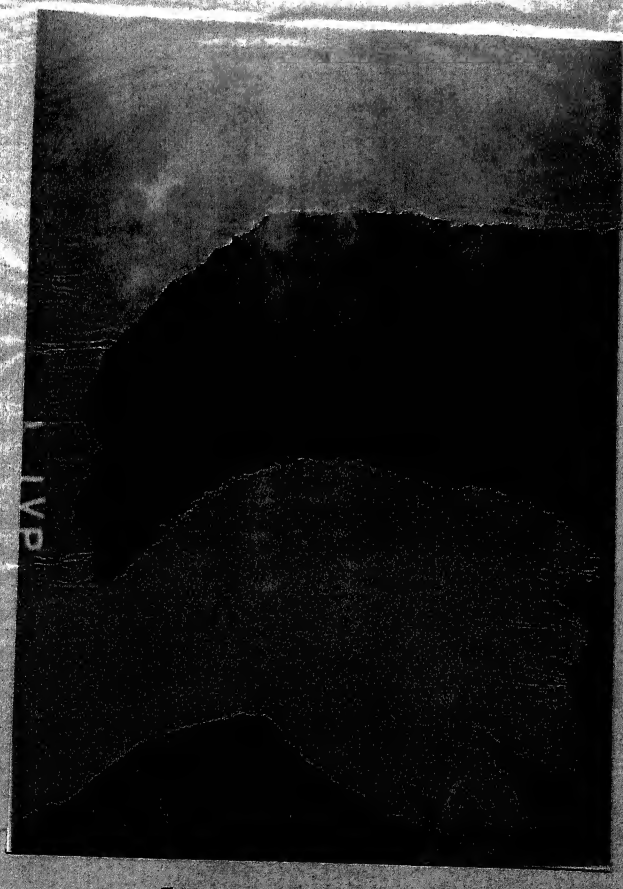


Photograph No.5
Vesical calculi showing spicules over the surface.

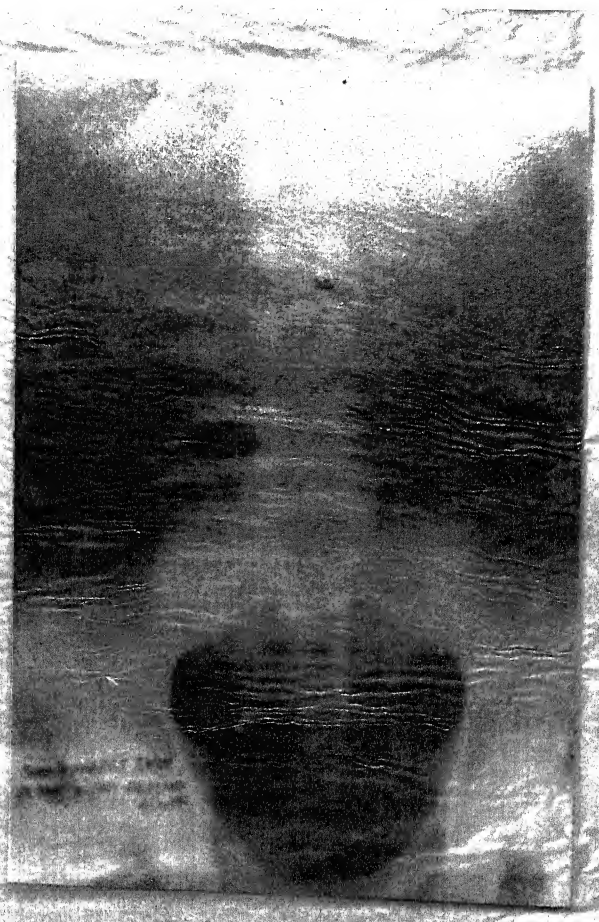
Photograph No.
Plain X-Ray abdomen showing left renal calculi



Photograph No.7
Plain X-Ray abdomen showing multiple renal calculi (right)

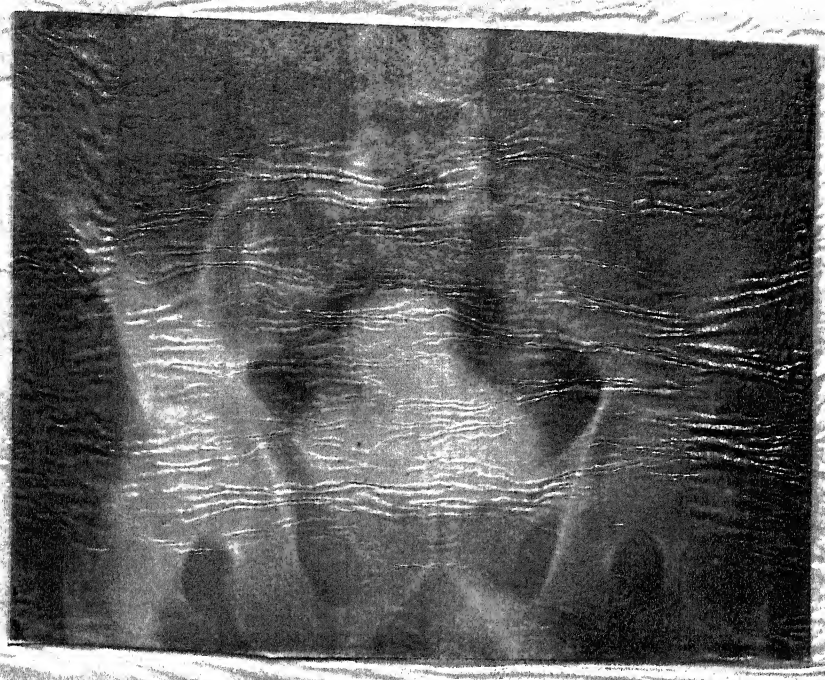


Photograph No. 8
I.V.P. showing left ureteric calculus
with functioning kidney on right side.

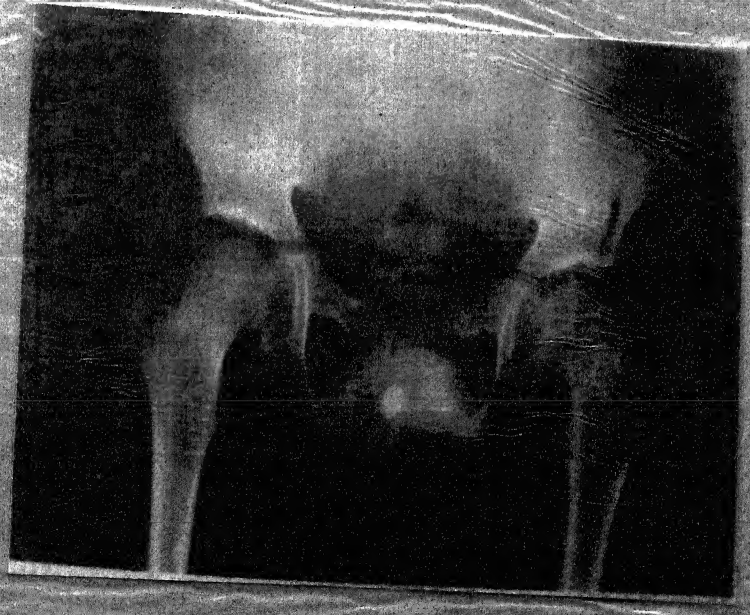


Photograph No.9
Plain X-Ray abdomen showing left ureteric Intramural calculus

Photograph No. 10
Plain X-Ray pelvis showing a large bladder calculus



Photograph No. 11
Plain X-Ray abdomen showing multiple bladder calculi



Photograph No. 12
Plain X-Ray pelvis showing urethral calculus.

D I S C U S S I O N

DISCUSSION

The present study was carried out in 50 cases suffering from urolithiasis in the surgery wards of MLB Medical College Hospital Jhansi during July 1993 to June 1994. Only Radiologically proved cases were included in this study.

After recording bio-data, clinical features were studied, aided by biochecmical methods, there interpretation, possible aetiological factors were identified, treatments, and analysis were cosidered.

INCIDENCE

Our Study Figures :- (Table No.1)

The incidence of urolithiasis was found to be about 124 per 10,000 hospital admissions during the above time span. This was 6.24% of the total admissions in surgical wards.

Previous Study Figures :--

Lower incidence was reported by Kabra (1972), 3 to 4% of all admitted general surgical cases in south eastern Rajasthan. Colabawalla (1970) reported, 50 per 10,000 hospital admissions, Nordin and Hodgkinson (1972) 1.91 (Sweden) to 19.21 (south corolina) per 10,000 admissions and Bailey (1974) 48 per 10,000 admissions at Christchurch Hospital.

Higher incidence reported by Curhan. G.C. et al (1991) - 13% in mid atlantic region of United States

and 31% in north west region of United States.

Much variations are encountered, as far as the incidence is concerned by different workers. The incidence in our series is higher as compared to other available reports. This is possibly due to peculiar climatic condition of prolonged high temperature and a long summer leading to low urinary output.

CLIMATIC AND SEASONAL FACTORS :--

Our Study Figures :- (Table No. 2)

Maximum admissions occurred (58%) in summer (June,July and August.).In this season maximum temperature ranged between 42-46 degree C.

Previous Study Figures :-

Prince and associates (1956, 1960), Rivera (1973), AL-Dabbagh and Fahadi (1977) and Elliott (1975) reported peak occurence in July, August and September. F Hussain (1990) from Bombay reported increase incidence of upper Urinary tract stone formation in Novemver in males. Yetcaddoff and co-workers found no seasonal variation.

Thus occurrence of urolithiasis, regarding climatic and seasonal variation in our series is similar to other available reports which is maximum in summer.

HEREDITY :--

Our Study Figures :- (Table No.9)

- One case (2%) having family history of stone
- Two cases (4%) having recurrent stones

Previous Study Figures :-

Resnick and Co-workers (1968) and white and colleagues (1969) accepted the hereditary theory.

Dretler etal (1969), Marquardt (1973) and Giugliani (1985) reported hereditary role in urolithiasis. Sutherland and colleagues (1985) reported risk of re-ccurence at age of 1.5 and 8 year. Trinchieri A et al (1988) observed higher frequency of stones amongst the first degree relatives. In his series family history of renal stone 45% in males and 31% in females were present.

Various reports as in our study show relationship between urolithiasis and heredity.

LOCATION

Our Study Figures		Previous study figures	
Table No. (3)		Tyagi etal (1974)	F.Marickar(1977)
Renal Calculi -	42%	22.35%	35%
	52%	28.24%	58%
Ureteric Calculi -	10%	5.89%	23%
Vesical Calculi -	44%	65.88%	24%
	48%	68.23%	23%
Urethral Calculi -	4%	2.35%	3%
Spontaneously passed -	-	3.53%	15%

F.Hussain et al (1990) reported ratio of UUT to LUT calculi cases 1.3. Aurora, Taneja and Gupta et al (1964) reported 62% vesical calculi cases.

Much variations are encountered regarding the occurrence of urolithiasis as per location by different

workers. Which are not similar with our study figures. In our study figures the ratio of UUT to LUT calculi is allmost equal. It is difficult to explain the reasons for the discrepancy.

AGE :--

Age incidence in UUT calculi cases :--

Our Study Figures :- (Table No. 4)

Maximum cases(65.38%) of UUT Calculi are in second and third decade of life.

Previous study figures :-

The age span in series of Das (1971), was between 21 to 50 year. Kabra et al (1974) noted maximum figures in second and fourth decade, shah and Julimdhwala (1959), Mehdiratta (1971) and F.Hussain (1990) noted miximum incidence in second and third decade of life.

Age incidence in LUT calculi cases :--

Our study figures :- (Table No. 4)

Maximum (54.16%) cases are in first decade of life.

Previous study figures :-

Yelloly (1830) noted 15% cases, Aurora et al (1963-64) more than 50% cases, Kabra et al (1972) 58% , and Hazarika et al (1974) 33% of LUT Calculi cases in first decade of life.

F.Hussain (1990) reported maximum cases of vesical calculus in first decade of life.

It is thus obvious that LUT calculi cases occur primarily in children (Ist decade of life) and UUT

calculi cases in adults (2nd to 5th decade of life). Our results tally with other available reports.

SEX :--

Our Study Figures :- (Table No. 5)

In UUT calculi cases male to famale ratio is 3:1

In LUT cases male to female ratio is 5:1

Previous Study figures :-

2:1 male to female ratio observed by Sriboonlue P et al (1992).

3:1 male to female ratio observed by Blacklock (1969), Fetler and Zimskind (1961), Inada et al (1958), Pak (1987), and Mehdiratta (1972).

5:1 by F.Hussain (1990) and 8:1 by Das (1971) and Halsted (1961). Nimikin (1992) reported male 54% and female 46% in his series.

Thus all the reported series agree on one point that males are more prone to calculus formation as compared to females as in our series. This may be due to oestrogen and progestrone hormones.

RURAL/URBAN POPULATION :--

Our Study Figures :- (Table No.6)

UUT calculi in rural population	30.76%
UUT calculi in urban population	69.24%
LUT calculi in rural population	70.83%
LUT calculi in urban population	29.17%

Various investigators reported that LUT calculi cases are more common in rural population and UUT calculi cases in urban population, our study figures

showes result similar to the previous ones.

RELIGION :--

Our Study Figures :- (Table No. 7)

Hindus - UUT calculi cases - 96.15%

LUT calculi cases - 91.66%

Muslims - UUT calculi cases - 3.85%

LUT calculi cases - 8.44%

Hindu to Muslim ratio is - 15:1

Previous Study Figures :-

Pandey (1979) reported Hindu to Muslim ratio 12:1, Shah et al (1959) 18:1 and Vashi (1959) 10:1.

Urinary tract calculi are more common in Hindu in comparision to Muslim, these finding are similar to other available reports.

DIET :--

Our Study Figures :- (Table No. 8)

	UUT calculi cases	LUT calculi cases
Vegetarian	- 53.85%	75%
Non vegetarians	- 46.15%	25%

Previous Study Figures :-

Gupta et al (1975) reported vegetarians 54% and non-vegetarians 46%.

Above figures show almost equal occurrence of UUT calculi cases in both vegetarian and nonvegetarian while predominance of LUT calculi cases in nonvegetarians

This inference has clear coincidence with others reported series. It may be the animal protein which is responsible for higher incidence of UUT calculi in non-

vegetarians.

CLINICAL FEATURES :--

Our Study Figures :- (Table No. 9 and 10)

	UUT calculi cases	LUT calculi cases
Pain in abdomen -	100%	100%
Heamaturia -	23.07%	16.66%
Burning in micturiton -	76.92%	66.66%
Pyrexia -	19.23%	16.66%
Sign of Urinary infection -	38.46%	45.83%
Anaemia -	-	16.66%
Malnutrition -	7.69%	33.33%
Renal Lump -	7.69%	-
Distended UB -	-	8.33%
Palpable urethral calculi -		8.33%

In UUT calculi cases pain was dull ache in renal cases (80%), colicky in nature and radiating loin to groin in ureteric cases (20%). In LUT calculi cases, pain was evident at the time of micturition in hypogastrium, perineum and radiating to the tip of penis.

Almost similar results reported by Bailey et al (1974) who observed renal pain in 85%,haematuria in 25% , Wani et al (1970) , pain in 76% , haematuria in 17%, renal angle tenderness in 14% and F Maricker (1977) pain in 97% but haematuria in 66% cases which was higher than present figures. Higgins (1939) reported that only 56% cases gave fairly typical history of an attack of colic.

INVESTIGATION :--

Our study figures :- (Table No. 12)

Urine Examinations :--

Albuminuria		20%
Urinary pH	acidic	66%
	alkaline	34%

Specific Gravity :

Normal	100%
Pus Cells	60%
RBC;s	22%
Crystal's	36%
Casts	18%

Urine culture and Senstivity (Table No.13)

UUT calculi	Sterile	E-coli	Klebsiella	Pseudomonas	Mixed
cases	76.92%	11.53%	-	3.85%	7.70%
LUT Calculi cases	37.50%	33.33%	4.16%	8.33%	16.66%

The urine of the patients showing crystals microscopically, were mainly of oxalate or phosphate type.

Previous study figures :-

These study figures were in accordance with those of Reza Gharib (1970) , Bailey (1974) , F Marickar (1977) and Wani (1976).

Haemoglobin was less than 10gm.% in 8% cases and between 10 to 14gm.% in 78.21% cases.

Leukcytosis was found in 8% cases. Signifi-

cant rise in ESR was present in 6% cases.

Blood were more than 40gm% in 6% cases mainly in UUT calculi cases. This is not in concordance with other studies which reported raised blood urea (>40mg.%) in 31% cases of Bailey) 1974), in 35% cases of F Marickar) 1977) and in 45% cases of Reza Gharib(1970).

PLANE X-RAY ABDOMEN SHOWING KUBP REGION :--

Our study figures :- (Table No. 14)

Renal - 81% cases having unilateral stones,(70% Right side and 30% left side).

- 19% cases having bilataral stones.
- 57% cases having single renal stones.
- 43% cases having multiple stones.

Ureter -

- 80% cases having unilataral stones (25% right
- side and 75% left side).
- one case showing staghorn calculi (2%) .

Previous study figures :-

Higgins (1939) noted multiple ureteral calculi . Braasch and Moore (1950) observed 1.7% bilatral stones. Kretschmer(1942) noted - 45.8% right side and 51.8% left side ureteral calculi. Drach et al (1986) noted 45% right side ureteral calculi , Segura et al (1985) noted 55% left side ureteric calculi and F Marickar (1977) reported 8% bilataral renal and 1% bilataral ureteric calculi.

On comparision of the findings of plain x-ray

abdomen showing KUBP region it is noted that there is significant discongruity between the present study and previous ones.

IVP :--

Our study figures :- (Table No. 15)

- Delayed excretion with hydronephrosis - 8% cases.
- Delayed excretion with out hydronephrosis- 8% cases.
- Non functioning kidney - 2% cases
- Ectopic kidney (Horse-shoe shaped) - 2% cases.
- Normal excretion 56% cases

Rest patients were subjected for USG examination showing normal renal and pelvicaleceal system with calculi.

Previous study figures :-

A relatively higher incidence of non-functioning kidney were reported in the previous studies conducted by Wani (1976) and Fazil Marickar (1977) who gave the figures of 11% and 23% respectively Delay in the appearance of the dye was also pointed out by Lalli (1974) and Van Assdalen and colleagues (1990)

SERUM ANALYSIS :--

Our study figures:- (Table No. 16 & 17)

Serum calcium was found to be with in normal acceptable limits of 8-11mg% in all cases. Hyperphosphataemia was found in 7.7% of UUT calculi cases and 8.33% of LUT calculi cases . In rest of the cases normophosphataemia was evident.

Previous study figures :-

On the contrary studies showed higher incidence of hypercalcaemia, 29% cases (Fazil Marickar 1977), 23% cases (Wani et al 1976) and 35% cases (Andersen 1962). However a lower incidence of hypercalcaemia was reorted by Bailey (1974) in only 4% cases. Similarly hypophosphataemia was reported by Bailey (1974) and F Marickar (1977) in 25% of upper and 35% of LUT calculi cases respectively. However Andersen (1962) reported normal serum inorganic phosphorus level in all his patients.

MANAGEMENT :--

All patients were subjected to operative modalities for treatment.

Our study figures (Table No. 18)		Previous study figur F-Marickar (1977)	Raza Gharib (1970)
Pyelolithotomy-	4% cases	20%	11.52%
Nephrolithotomy -	36% cases	7%	--
Nephrectomy -	2% cases	4%	2.42%
Nephrostomy -	--	4%	2.42%
Ureterolithotomy -	10% cases	8%	5.44%
Cystolithotomy -	44% cases	19%	76.97%
Urethrolithotomy -	4% cases.	--	.06%
Meatotomy -	--	1%	--

The above results do not resemble with those of the modality of treatment offered by F Marickar (1977)

and RezaGharib (1970). Cystolithotomy was performed in fewer cases by F Marickar (1977) due to less nummber of bladder calculi cases in his series.

Ureterolithotomy was performed in less number of cases by Reza Gharib (1970) due to lower percentege of ureteric calculi cases in his series.

ANALYSIS OF URINARY CALCULI :--

Analysis of urinary calculi was carried out to find the chemical composition of stones. In the present series, Weight of urinary calculi ranged between .279 to 96.059gms. This observation is not in resemblance with the series of Thind and Nath (1969), Tyagi et al(1974) and Kabra et al (1976). They reported the weight of urinary calculi to be from 0.016 gms to 22gms., 0.2gms to 60 gms, in their respective series. The calculi are heavier in the patients of Bundelkhand area. The shape, surface and internal structure of all the calculi are similar to what was reported in other series in India.

MOISTURE CONTENT :- (Table No. 19)

All sample of our seriese are having moisture. In UUT calculi cases maximum moisture content lies in range of 6-10mg% in 46.15% cases and in LUT calculi in 50% cases.

ASH CONTENT :--

Ash is present in all stones of our series. In UUT calculi cases maximum ash content lies in range of 51-75mg% in 57.70% cases and in LUT calculi in 41.67% cases. Ash content more than 76mg% present in LUT calcu-

li in 37.50% cases.

CHEMICAL COMPOSITION OF UROLITHS :--

Our study figures :-

In the present study,(Table No. 21,22,23,24 and 25) calcium, phosphate, magnesium, oxalate and cholestrol was present in all cases. Thus in our study all the stones are of mixed type.

Previous study figures :-

Summarizing the observations of number of investigators (Table No. 26) of the world today(Kampbell) the commonest types of calculi are calcium oxalate, calcium phosphate or mixtures of the two. The relative incidence of uric acid and cystine calculi stays approximately the same the world over. But in India the relative incidence of uric acid calculi is less. The mixed urinary calculi as can be seen from Table No. 26 are also very commen. It seems that through out the world, approximately the same percentage of population is affected with a given type of urinary lithiasis.

Also summarizing (Table No. 27) the comparative study of incidence of different constituent of urinary calculi in four geographical area of India, reported by various authors, including :

Northern area - Chandigarh

Central area - Delhi, Gwalior and Jhansi

Western area - Bombay, Rajasthan, Ahamdabad and Ahamad Nagar.

Southern area - Kerala and Trivandram.

The calcium is present in almost all cases of urinary calculi in all these areas. The Calcium, Oxalate, Phosphate, Urate and Magnesium are present in decreasing order in all these areas, except in Southern area (Kerala & Trivandram) and Ahamad Nagar, where magnesium is more than Urate.

The commoenest stone found in all these areas are calcium phosphate and calcium oxalate or mixture of two (mixed type). Urate stone became comparatively less in southern area (Kerala & Trivandram) as compared to Northern area (Chandigarh) and Central area (New Delhi, Gwalior and Jhansi).

It also seems that magnesium, Ammonium, Phosphate stones show an increase in relative incidence in southern area (Kerala & Trivendram) and in Western area (Bombay, Rajasthan and Ahamdabad) in comparision to northern (Chandigarh) and Central areas (Delhi, Gwalior and Jhansi).

The incidence of stone having carbonate is greater in western area (Bombay, Rajasthan & Ahamdabad), in comarision to central area (Delhi, Gwalior and Jhansi).

The relative incidence of fibrin, cystine and cholesterol is less in all these areas in comparision to calcium, phosphate and oxalate.

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SUMMARY & CONCLUSION

SUMMARY AND CONCLUSION

- Hospital incidence of urolithiasis in admitted patient is 6.24%.

- Maximum admissions (58%) occured in months of summer (June, July and Aug.). In this season maximum temperature ranged in between 42 - 46 degree C.

- Family history of urinary calculi is found in 1 case (2%) only and history of recurrent stones found in two cases only. 26 cases of UUT calculi and 24 cases of LUT calculi admitted at M.L.B. Medical College Hospital, Jhansi during 1993 and 1994 were studied in the present series. The ratio of incidence of UUT to LUT calculi is almost equal. Incidence of renal calculi is 42%, ureteric calculi 10%, bladder calculi 44%, and urethral calculi 2% out of the total urinary calculi cases. The incidence of bladder calculi was significantly low in developed countries like Europe and America but the incidence is high in our series as will as in those reported by other Indian workers.

- The commonest age group observed in this series for upper urinary tract calculi is 11-30 years (65.38%) and for lower urinary tract calculi is 0-10 years (54.16%).

- The ratio of male to female is 3:1 in upper urinary tract calculi and 5:1 in lower urinary tract calculi cases. Thus males were more involved in urinary calculi disease.

- The incidence of upper urinary tract calculi is more in urban area (69.24%) and of lower urinary tract calculi in rural area (70.83%).

- Hindus dominated the list both in upper urinary tract calculi (96.15%), as well as in lower urinary tract calculi (84.61%) . Hindu to Muslim ratio is 15:1.

- Incidence of upper urinary tract caculi in vegetarians and non-vegetarians is more or less the same. 53.85% cases are vegetarians and 46.15% are non-vegetarians. Lower urinary tract calculi are more common in vegetarians (75%).

- The cardinal features of upper urinary tract calculi are pain in abdomen (100%), either colicky or dull ache-type, haematuria (23.07%), burning in micturition (76.92%) and fever (19.23%) while pain (100%) , either hypogastric or perineal, increased frequency of micturition and burning in micturition (66.66%) are the chief features of lower urinery tract caculi. Associated diseases with the urinary caculi are benign prostatic hyperplasia (4cases), tuberculosis lung (3cases).

- Hb is < 10gm % in four cases (8%) and leucocytosis in four cases (8%). Albumin is present in the urine of 10 patients and microscopic haematuria is evident in 11 patients, pus cells in 30 cases, crystals in 18 and cast in 9 cases. Urine culture demonstrated bacteria in 6 cases (23.07%) of upper urinary tract caculi and 15 cases (62.50%) of lower urinary tract calculi. Blood urea level is high in 3 cases (6%) mainly of upper urinary tract

calculi and normal blood sugar in all patients.

- Plane X-Ray abdomen, 21 cases showing renal stones of which 17 cases having unilatrel and four cases having bilatrel stones,5 cases showing ureteric stones, 22 cases UB and 2 cases of stones in urethra. In 2 cases, intravenous pyelograpahy revealed evidences of hydronephrosis . One case has non-functioning kidney on the affected side and one has ectopic kidney (Horse shoe shape). Multiple renal calculi are found in 9 cases and staghorn calculus in one case. Rest of the patients showing normal U.S.G.

- Serum analysis revealed normal serum calcium level in all Patients while hyperphosphataemia are detected in 2 cases (7.75%) of upper urinary tract calculi and 2 cases (8.33%) of lower urinary tract calculi.

- Pyelolithotomy, Nephrolithotomy and Ureterolithotomy are performed in uncomplicated cases of upper urinary tract calculi, nephrectomy in 1 case (staghorn calculus) of non functioning kidney. Cystolithotomy in 22 cases and urethero lithotomy in 2 cases are performed in LUT calculi cases.

- Urinary calculi of different size, shape, weight and colour are observed.

- All stones have moisture and ash content. In UUT calculi cases maximum moisture lies in range of 6 - 10 mg% in 46.15% cases and in LUT calculi cases in 50% cases. Maximum ash content lies in range of 51 - 75 mg% in UUT calculi cases and more than 76mg% in LUT clculi cases in 37.5% cases.

- Analysis of urinary stones reveal that all stones of our series are mixed type .

- Summarizing the reports avalaible by verious authors from all over India this concluded that most common stones present are of mixed type. Urate stones are comparatively less in Southern area of India and Magnesium Ammonium Phosphaste stones show so an relative increase in incidence in Western and Southern areas of India.

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PROFORMA OF CASE-SHEET

Name Age/Sex Occupation

Rural/Urban Address

D.O.A. D.O.D.

Diagnosis

CHIEF COMPLAINTS:

Pain

Frequency of micturition

Haematuria

Difficulty in micturition

Retention of urine

Burning in micturition

Fever

Other

H/O Present Illness in detail

Past History: H/o reccurent stone, D.M., H.T., C.H.F., features of secendary hyperparathyroidism, renal tuberculoiss and recumbency.

Family History: History of stone in family

Personal History:

Dietary History

Smoking

Addiction to any intoxicant

Clinical Examination:

Malnutrition Renal Lump

Anaemia

B.P.

R/R P/R

Signs of Uraemia

Signs of Urinary infection

Distended Bladder

Plpable Uretheral calculus

Palpable Bladder calculus

Palpable prostatic calculus

Systemic Examination:

Investigations:

Urine - Albumin Sugar

Microscopic -

RBC Puscells

Casts Crystals

Culture and Sensitivity

Specific gravity pH

Blood - TLC DLC - P L E M

ESR Hb%

Blood Urea Blood Sugar

Serum Calcium Serum Inorganic Phosphorus

Radiological - Plain X-ray abdomen for K.U.B.P. region -

I.V.P. (If required) -

U.S.G.

Operation:

Name –

Findings –

Physical and biochemical Analysis of stone:–

Follow Up: